Chapter 22
Cervical dizziness

Ting-Ya Chang

Neurological institute, Taichung Veterans General Hospital

Cervical dizziness (vertigo)

- A long-standing interdisciplinary debate
  - → various forms and pathomechanisms
  - → Definition? Pain or no pain?

- Somatosensory signals (musculotendinous receptors in the neck) → accurate kinesthetic feedback of head movements
  - The perception of head motion and self-motion during active locomotion by converging with vestibular and visual input.

The difficulty of clinical evaluation

1. Insufficient pathophysiological knowledge of function and multimodal interaction of the sensory signals from the neck afference.
2. The existing confusion about the concept of so-called cervical vertigo.

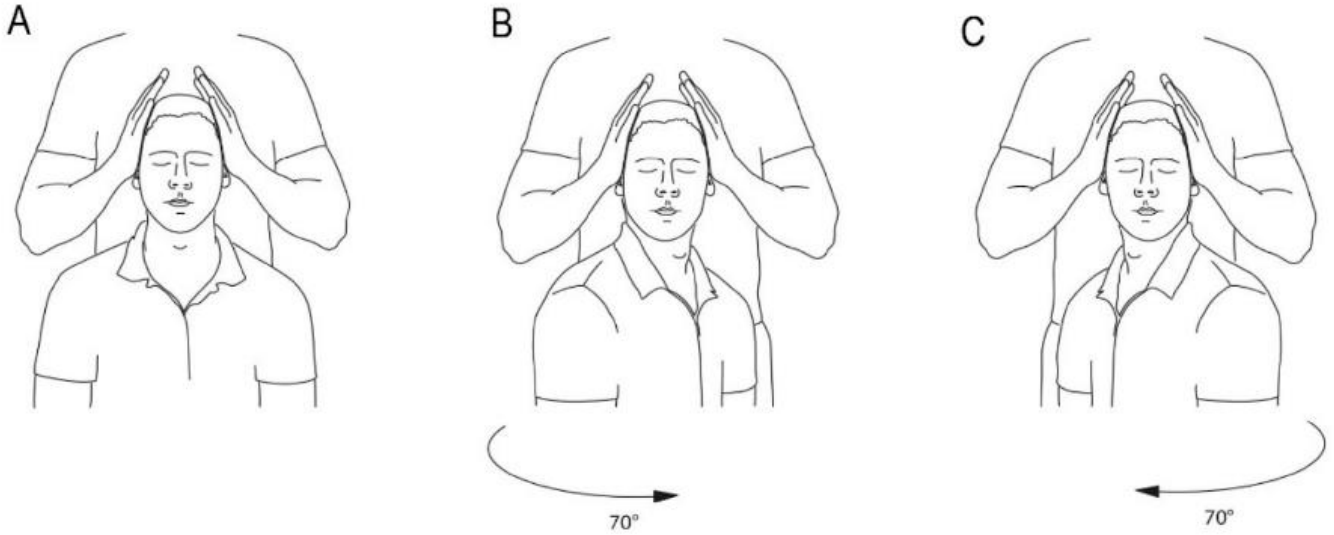
Evidence of cervical dizziness

- Neural connections between the neck receptors and the central vestibular system (the cervico-ocular reflex and the neck reflexes for postural control) have been investigated experimentally in **animals:**
  - Unilateral surgical deafferentation of C1 to C3 in the squirrel monkey & cat → locomotor ataxia
  - Local suboccipital anesthesia in the rhesus monkey → locomotor ataxia
- Local anesthesia of posture-lateral neck tissue in humans
  - ↑ ipsilateral and ↓ contralateral extensor muscle tone
  - → fall, gait deviation, and past pointing toward the anesthetized side
- *→ without convincing clinical relevance*

Evidence of cervical dizziness

- **Cervical headache** → unilateral anesthetic C2 neck blockades
  - Normal posturographic tests, SVV and EOM
  - Only small and transient disturbances of **ipsilateral gait deviation**
    - Minor contribution of the cervico-ocular reflex in subjects with an intact peripheral vestibular system
      - Gain of 0.1–0.2 as compared to 1.0 of the VOR
    - Vestibular sensitivity decreases (如 bilateral vestibulopathy), the COR becomes more sensitive and more important to compensate for the vestibular loss.

Clinical examination



- **Neck turning test**

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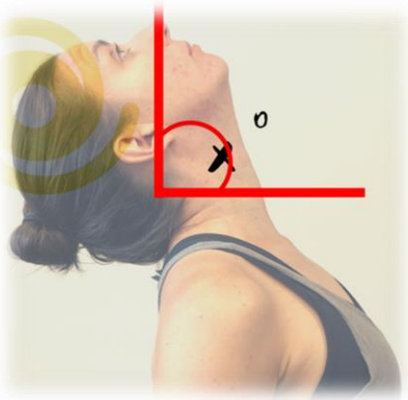
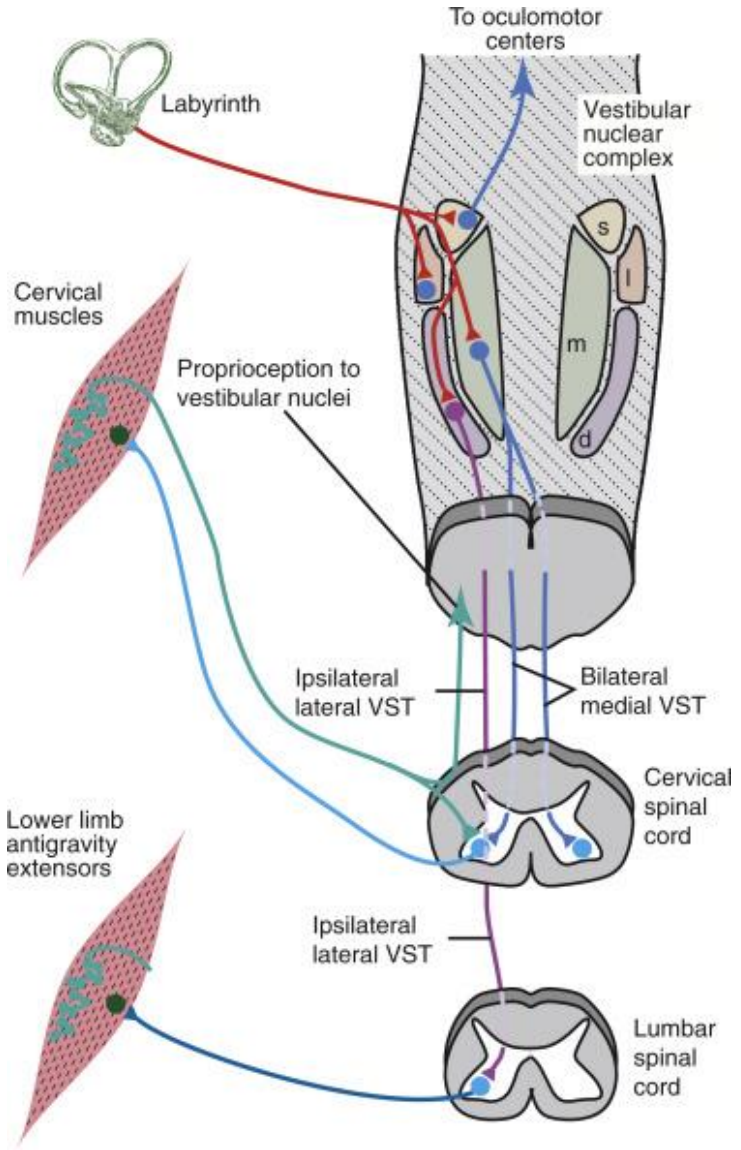
- **Static cervico-ocular reflex** or **Romberg's test** while leaning the head backward
  - Non-specific and insufficiently standardized
  - May induce nystagmus → but also in healthy controls



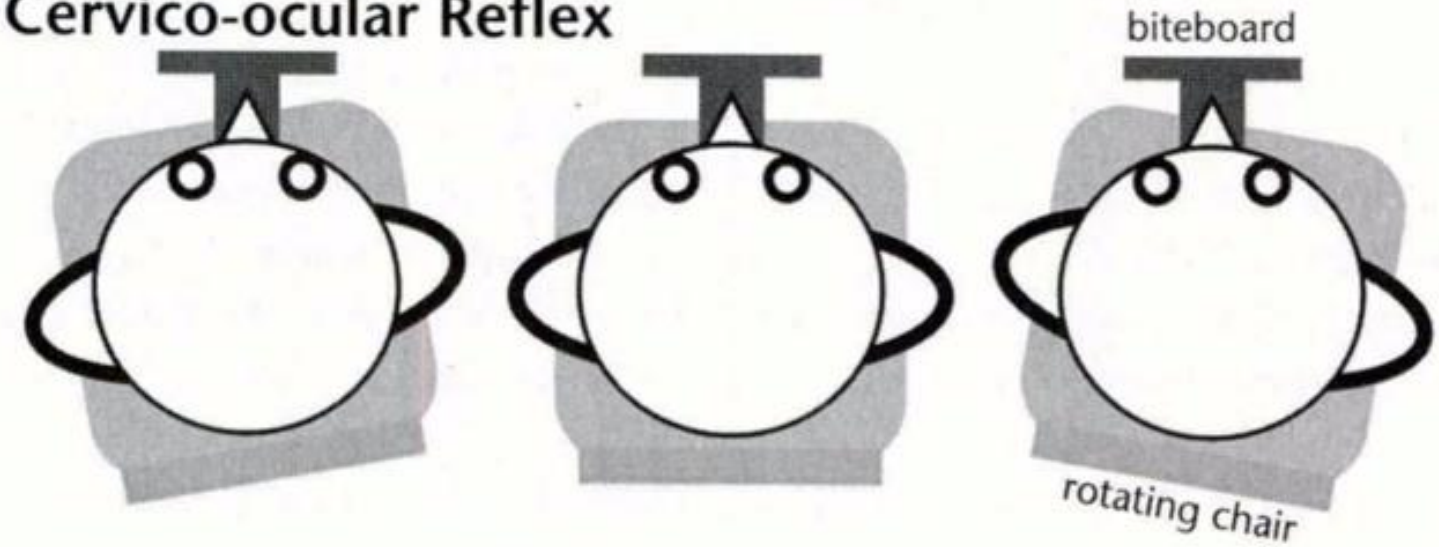
- ? "fantastic" successes by chiropractic treatment
  - → may represent a liberatory maneuver in patients with BPPV

Somatosensory signals of the neck

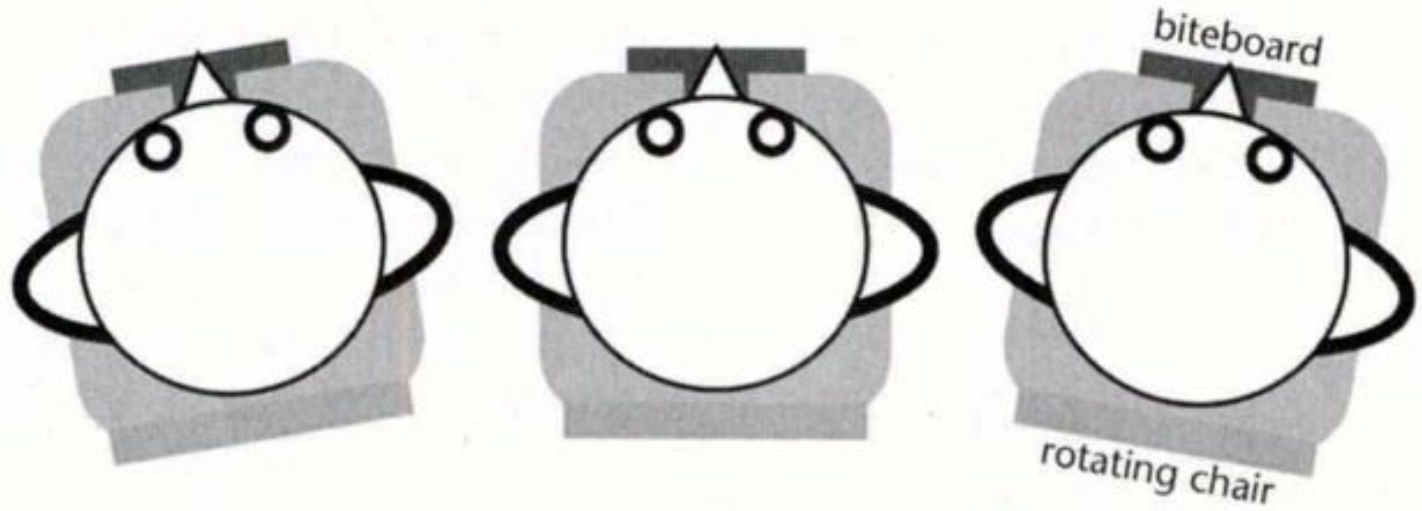
- Somatosensors in the muscles, joints, and skin
  - → induce self-motion and trigger nystagmus

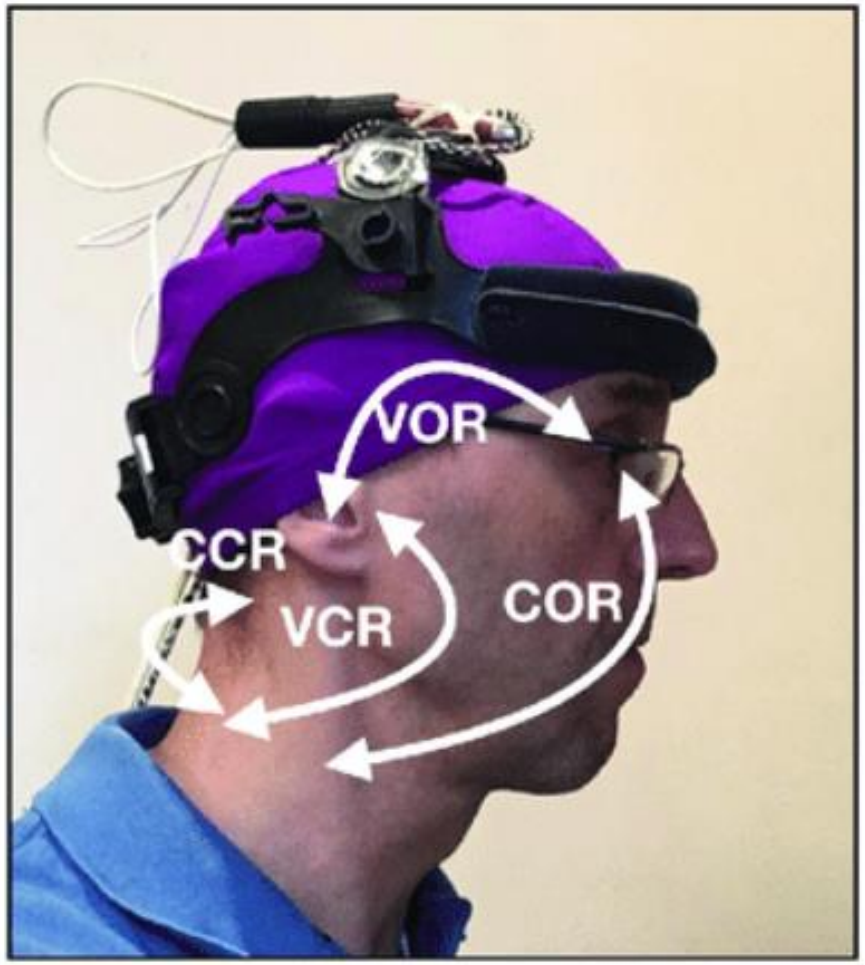


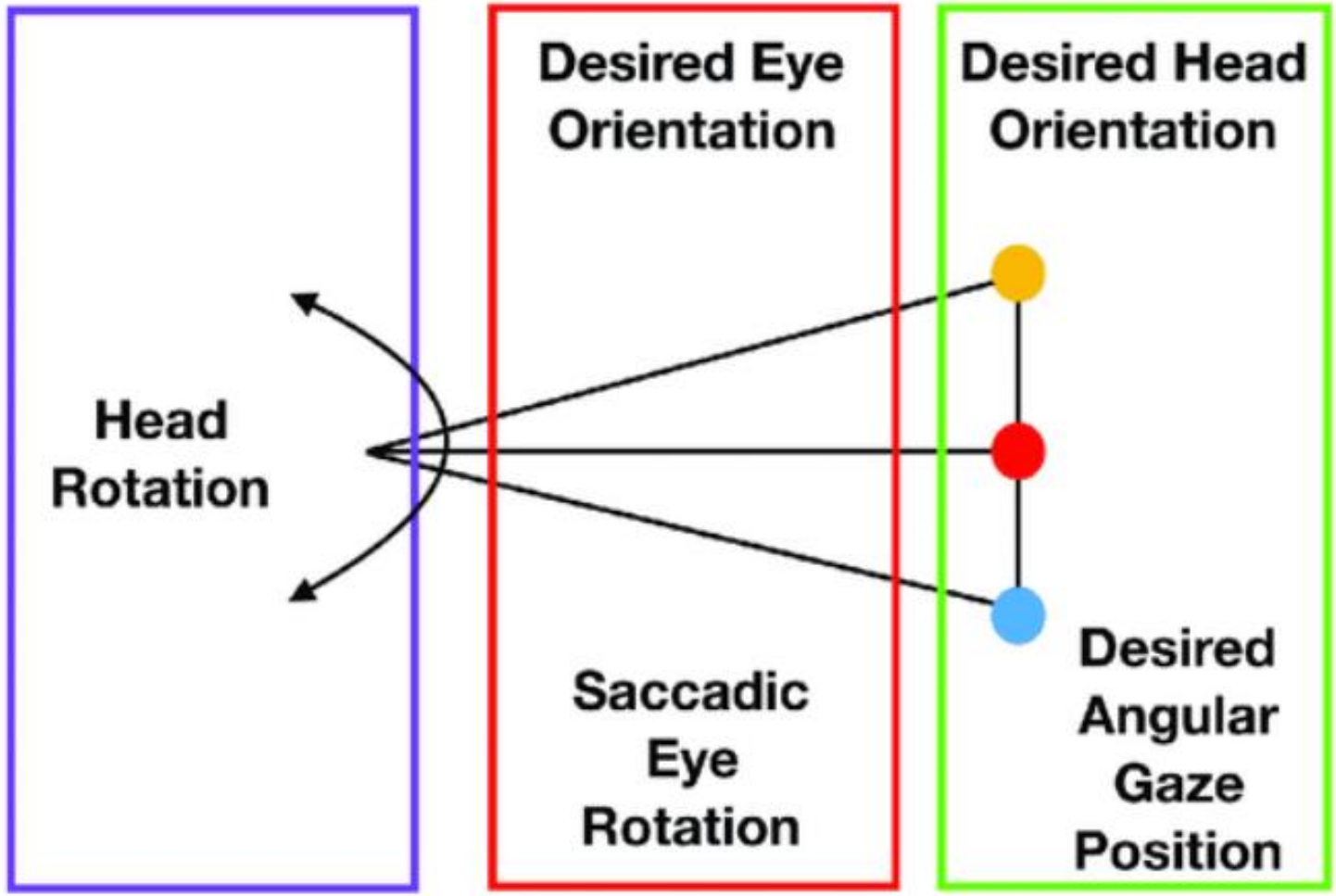
Cervico-ocular Reflex



Vestibulo-ocular Reflex

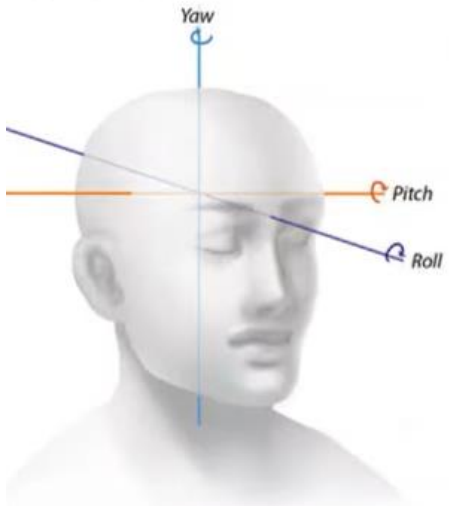






https://doi.org/10.1371/journal.pone.0219515.g006

Vestibulocolic and vestibulospinal reflexes correct the orientation of the head and body, respectively, when they are taken out of their normal, upright position. It is initiated by the vestibular system in the inner ear (Figure 1), which carries information on the position of the head to the vestibular nuclei of the brain stem via CNVIII. Medial and lateral vestibulospinal tracts descend from the vestibular nuclei and terminate on interneurons that activate the motor neurons of the neck and lower body, respectively (Figure 2).



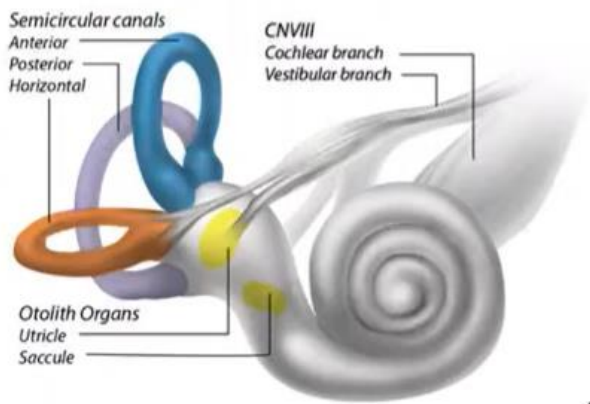


Figure 1. Vestibular appareatus and associated head movements (anterior view). Three semicircular canals detect rotational acceleration around 3 orthogonal axes: pitch, roll, and yaw. The saccule and utricle are otolith organs that detect linear acceleration in the horizontal and vertical planes.

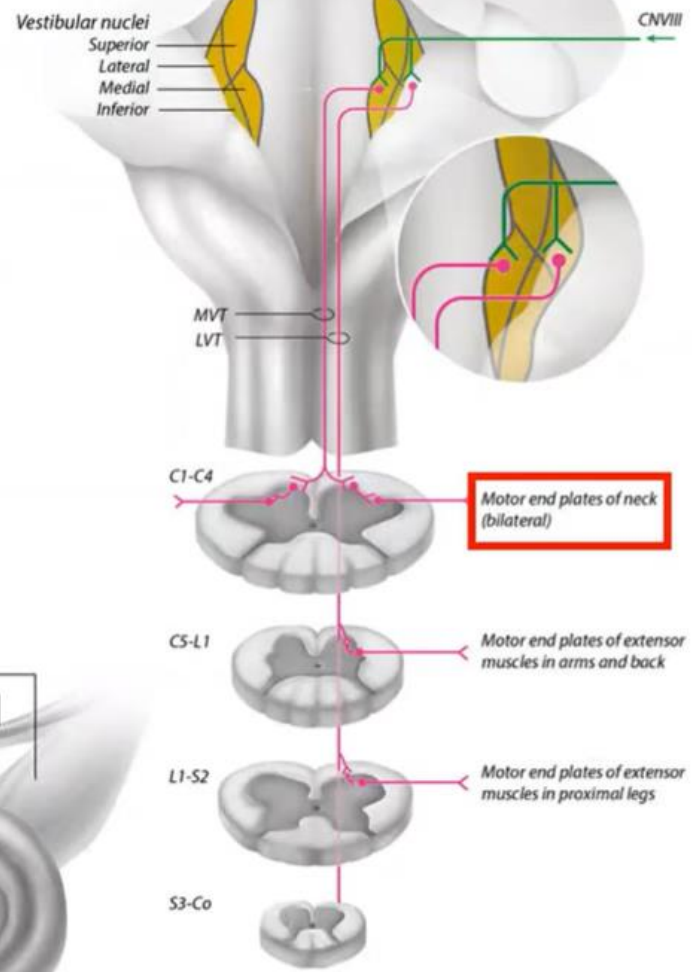


Figure 2. The vestibulospinal and vestibulocolic tracts through the brain stem and spinal cords (Posterior view). The the axons of the ipsilateral medial vestibular nuclei descend as the medial vestibulospinal tract (MVT) and are responsible for vestibulocolic reflexes that stabilize the head and neck. The lateral vestibular nuclei's axons descends as the lateral vestibulospinal tract (LVT) and are responsible for vestibulospinal reflexes that stabilize the limbs and trunk.

- **Vestibulocollic reflex (*VCR*)** | reflex that allows maintenance of balance when postural sway is detected through movement of neck muscles
  - **Sensory** | CN VIII
  - **Motor** | Cervical spinal nerves
  - **Purpose** | Maintain upright head

- **Vestibulospinal reflex (*VSR*)** | reflex that allows maintenance of balance when postural sway is detected through movement of trunk, arm, leg muscles
  - **Sensory** | CN VIII
  - **Motor** | Thoracic and lumbar spinal nerves
  - **Purpose** | initiate a hip or step strategy to maintain balance

https://www.youtube.com/watch?v=7XJRCfM19ZI

“ War between believers and doubters”

- The hypothetical neurophysiological explanation is still mainly only of theoretical significance.

Head Motion-Induced Cervical Split-Second Vertigo

22.1

Head Motion-Induced Cervical Split-Second Vertigo

- **Acute neck pain syndrome <--> Long-lasting spinning vertigo/balance disorder**
  - Restricted mobility of head vs. trunk movements
  - Rapid voluntary head movements
  - → cervical vertigo/dizziness or seemingly body pushs
  - → sometimes postural instability for fractions of a second

- Moderate clinical relevance
  - No particular treatment
  - Spontaneous remission within days

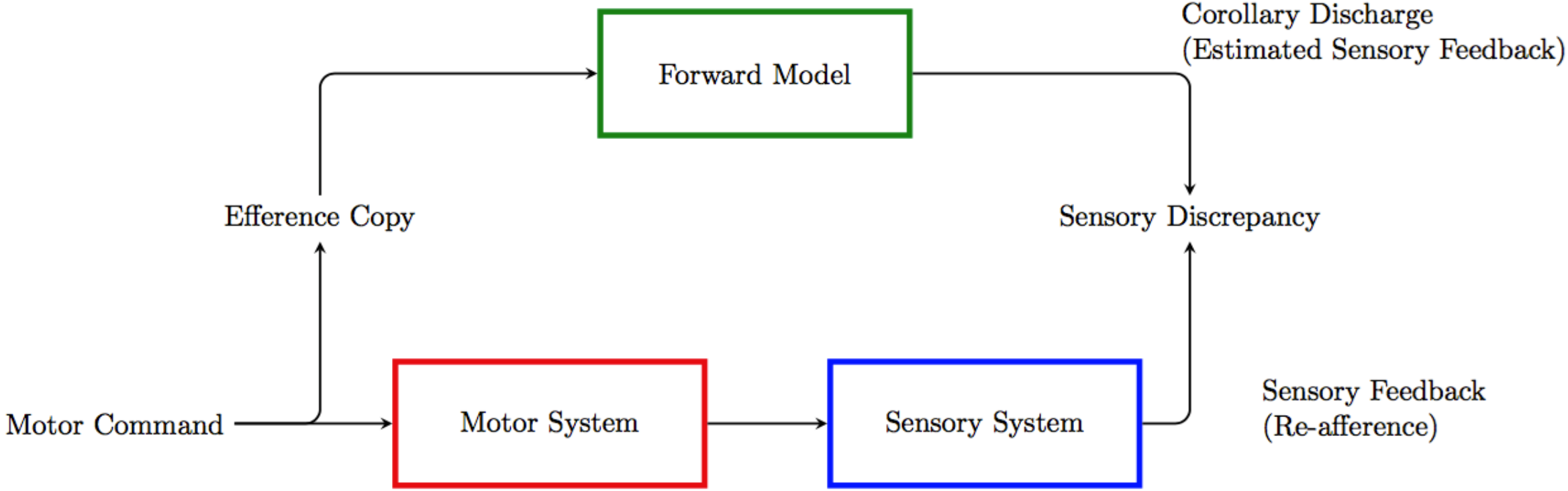
Head Motion-Induced Cervical Split-Second Vertigo

- Complaint of the patients:
  - “Apparently involuntary body perturbations or tilts”
  - “Apparent movements of the surroundings only elicited by rapid head movements that are hindered by a painful blocking of head rotations.”

- **Hypothetical mechanism**
  - The **efference copy** and the **reafference principle** → “internal model theory “
    - Expected reafference of an intended head rotation

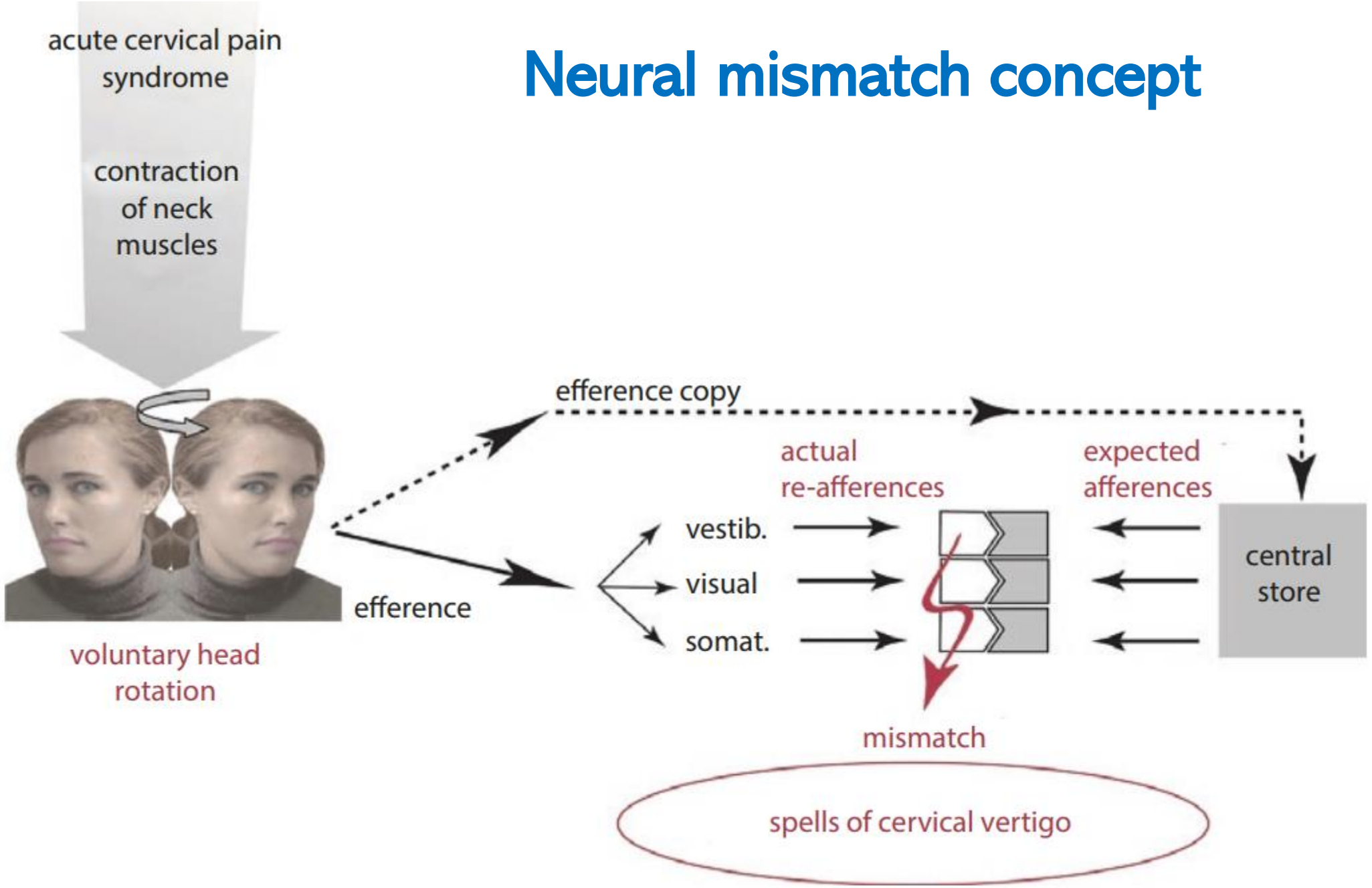
    <=/=> mismatch by the painful neck muscle stiffness

    - Sensory input calibrated by earlier experience of head motions
  - May gradually re-adjusted in chronic condition



Theory of Motor Control UMD

Neural mismatch concept



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DOI:10.3233/VES-220202
IOS Press

The Bárány Society position on 'Cervical Dizziness'

Terminology: prefer the use of "cervical dizziness"

- *Exclude cervical vascular vertigo and whiplash injury*

Conclusion

- *Given the current data, we cannot at present recommend any specific diagnostic criteria for cervical dizziness, nor can we presently recommend any specific therapy.*

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The Bárány Society position on 'Cervical Dizziness'

- **Consistent features**
  - 1. Neck stiffness and pain are aggravated during neck movements.
  - 2. Neck movements trigger transient imbalance and/or light-headedness and/or illusory self motion
  - 3. Neck-directed therapy improves neck pain, neck stiffness and dizziness.

- **Exclusion**
  - 1. There is no neck pain or discomfort.
  - 2. The dizziness ever occurs spontaneously, or if the dizziness is exclusively positional (i.e., when the head orientation with respect to gravity changes).

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The Bárány Society position on 'Cervical Dizziness'

Pathophysiology

- *The somatosensory input hypothesis*
- *Triggered migraine hypothesis*
- *Trigeminal hypothesis not invoking migraine*
- *Neurovascular hypotheses of Barr´e and Lieou*
- *Carotid sinus syndrome and associated syncope-mediated hypotheses*

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The Bárány Society position on 'Cervical Dizziness'

Pathophysiology

- *Triggered migraine hypothesis*
  - Pain inputs to trigeminal circuits → trigeminal nociceptive activation
  - Migraine→ secondary cervical stiffness

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The Bárány Society position on 'Cervical Dizziness'

Pathophysiology

- *Trigeminal hypothesis not invoking migraine*
  - Somatosensory hypothesis → cervical headache
    - Nociceptive afferents from the ophthalmic trigeminal division and from spinal nerves C1, C2 and C3, converge onto second-order neurones in the trigeminocervical nucleus
  - 147 patients with cervicogenic symptoms: dizziness, headache
    - Half of the patients reported improved dizziness with greater-occipital nerve injection

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The Bárány Society position on 'Cervical Dizziness'

Pathophysiology

- *Neurovascular hypotheses of Barr´e and Lieou*
  - *Barre' hypothesised (and subsequently independently by Lieou) that mechanical compression by cervical spine spondylosis of the sympathetic plexus that surrounds the vertebral arteries could trigger vertigo via vertebrobasilar constriction*
    - *Subsequent laboratory experiments in animals **could not find** evidence for this hypothesis*

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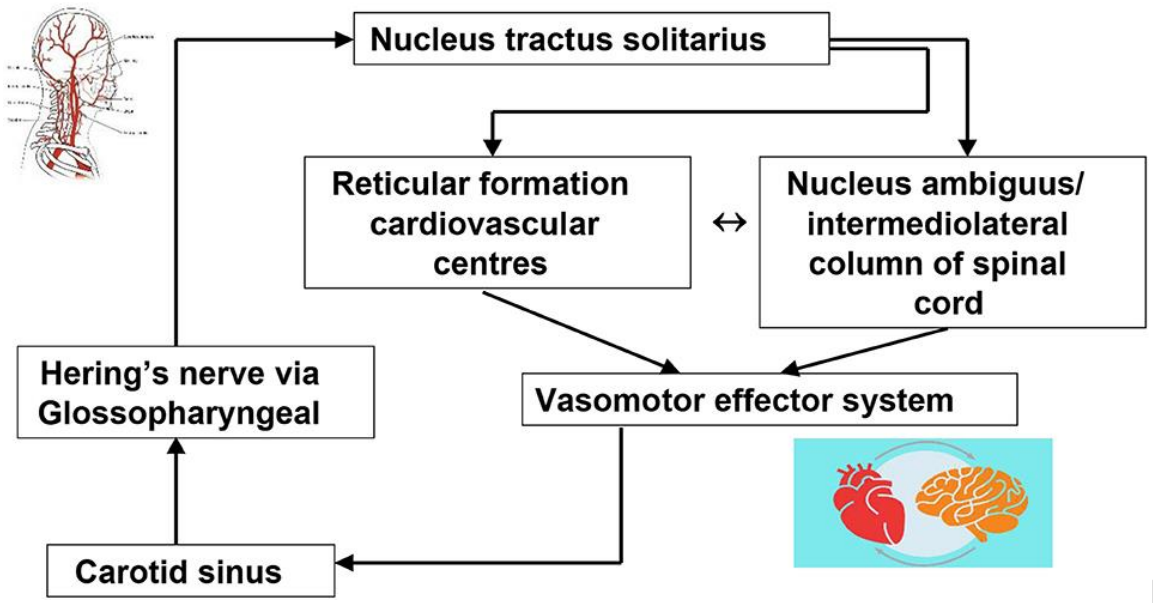
The Bárány Society position on 'Cervical Dizziness'

Pathophysiology

- *Carotid sinus syndrome and associated syncope-mediated hypotheses*

Carotid baroreflex neuroanatomy



Front. Cardiovasc. Med., 22 April 2020

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The Bárány Society position on 'Cervical Dizziness'

Pathophysiology

- *Carotid sinus syndrome and associated syncope-mediated hypotheses*
  - Rapid head turn could trigger both neck pain and transient bradycardia and hypotension → light headedness and presyncopal symptoms
  - Animals study: neck afferents modulate cardiovascular reflexes to a modest extent (cardio-inhibitory response)
  - Requiring more studies.

Vertebral Artery Compression/Occlusion Syndrome

22.2

Vertebral Artery Compression/Occlusion Syndrome

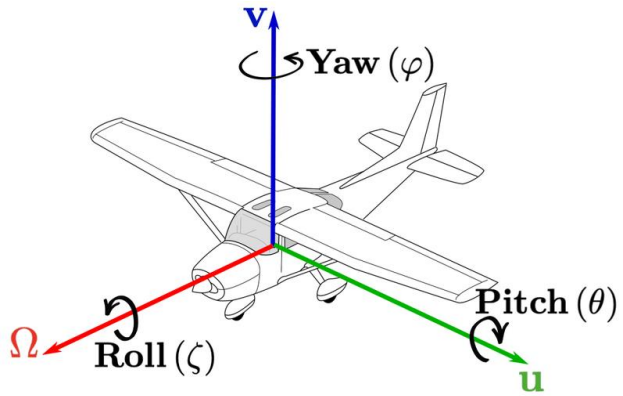
- Rare but clinically most relevant and well-defined vascular form of cervical vertigo
  - Head rotation→ symptomatic compression/occlusion of the dominant VA
- = ***Bow Hunter's syndrome***

Key:

- 1. Initial symptoms: spinning vertigo and nystagmus elicited by head rotation to one side in the yaw plane.
- 2. There is a danger of impending, life-threatening vertebrobasilar infarcts.
- 3. It can be efficiently cured by surgical decompression in the majority of cases.

- Stenosis or vessel malformation
  - VA hypoplasia or termination in the PICA
  - A “dominant” contralateral VA

- Head rotation toward the non-dominant VA
  - → Compress the dominant VA (usually at the atlantoaxial C1 - C2 level)
    - Muscular and tendinous insertions, osteophytes, and degenerative changes
    - Aneurysms or arterial dissections (less frequent)



Clinical manifestation

Key symptom:

- A severe **rotational vertigo** elicited by a prolonged horizontal head rotation → rapidly abates when the head is rotated back to its normal position.

- **Down-beating nystagmus**
  - Ischemic dysfunction of the brainstem/caudal cerebellar midline structures

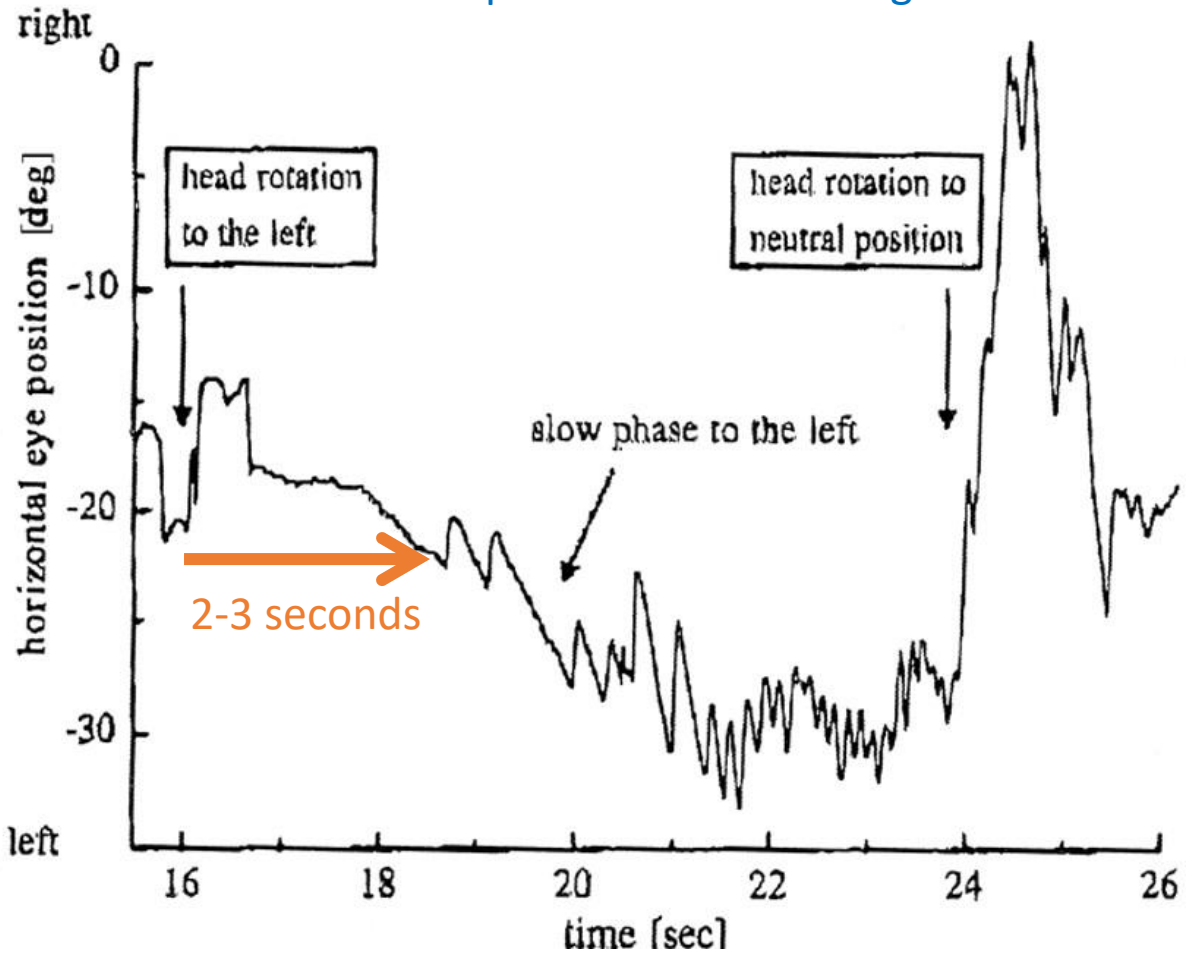
Case report

- 48-year-old man with severe rotatory vertigo for 2 years.
  - Head rotations to the left reproducibly elicited the attacks, which occurred 2 to 3 seconds after rotation.
  - Associated with a deep tone in the right ear (tinnitus).
  - Symptoms ceased promptly after turning his head to the neutral position with no additional symptoms
  - Normal neurologic examination during the attack.

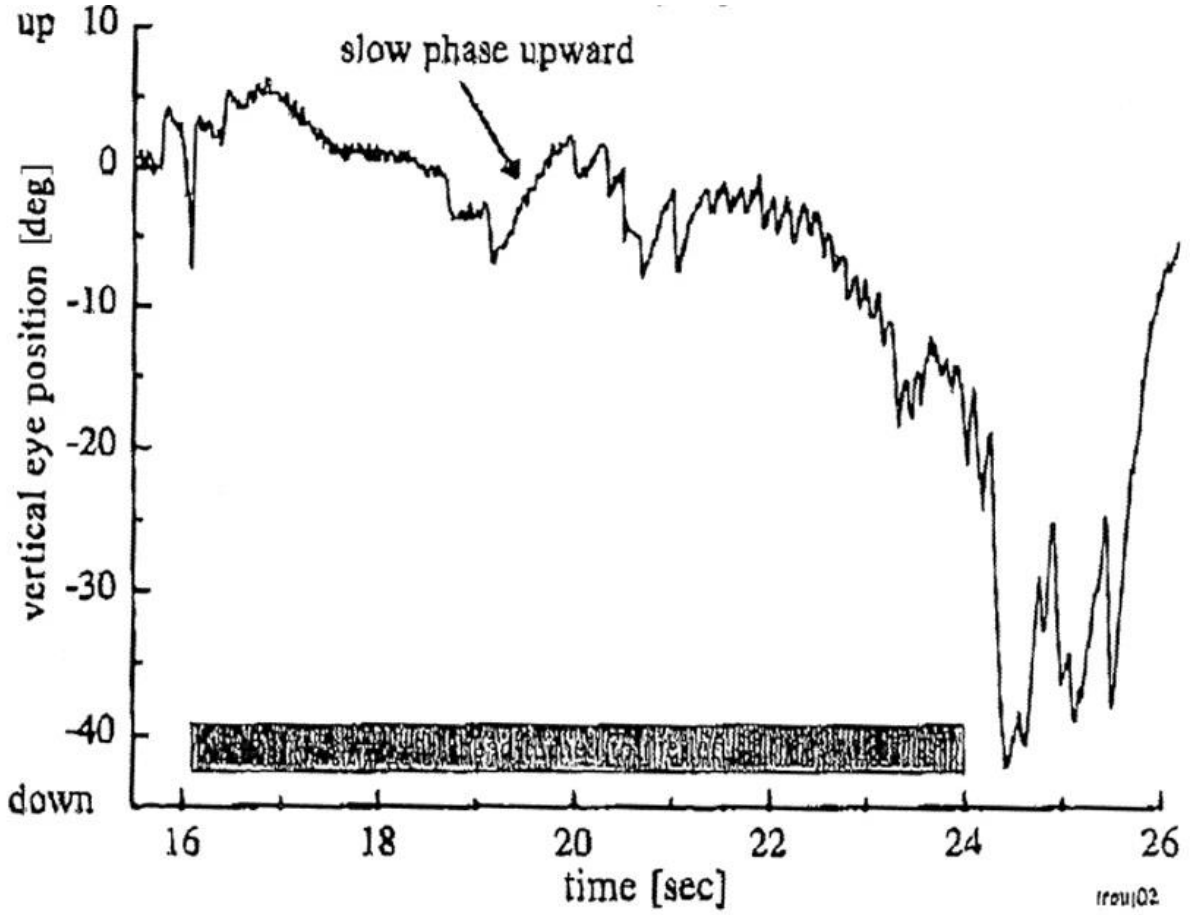
M Strupp et al. Neurology. 2000 Mar 28;54(6):1376-9.

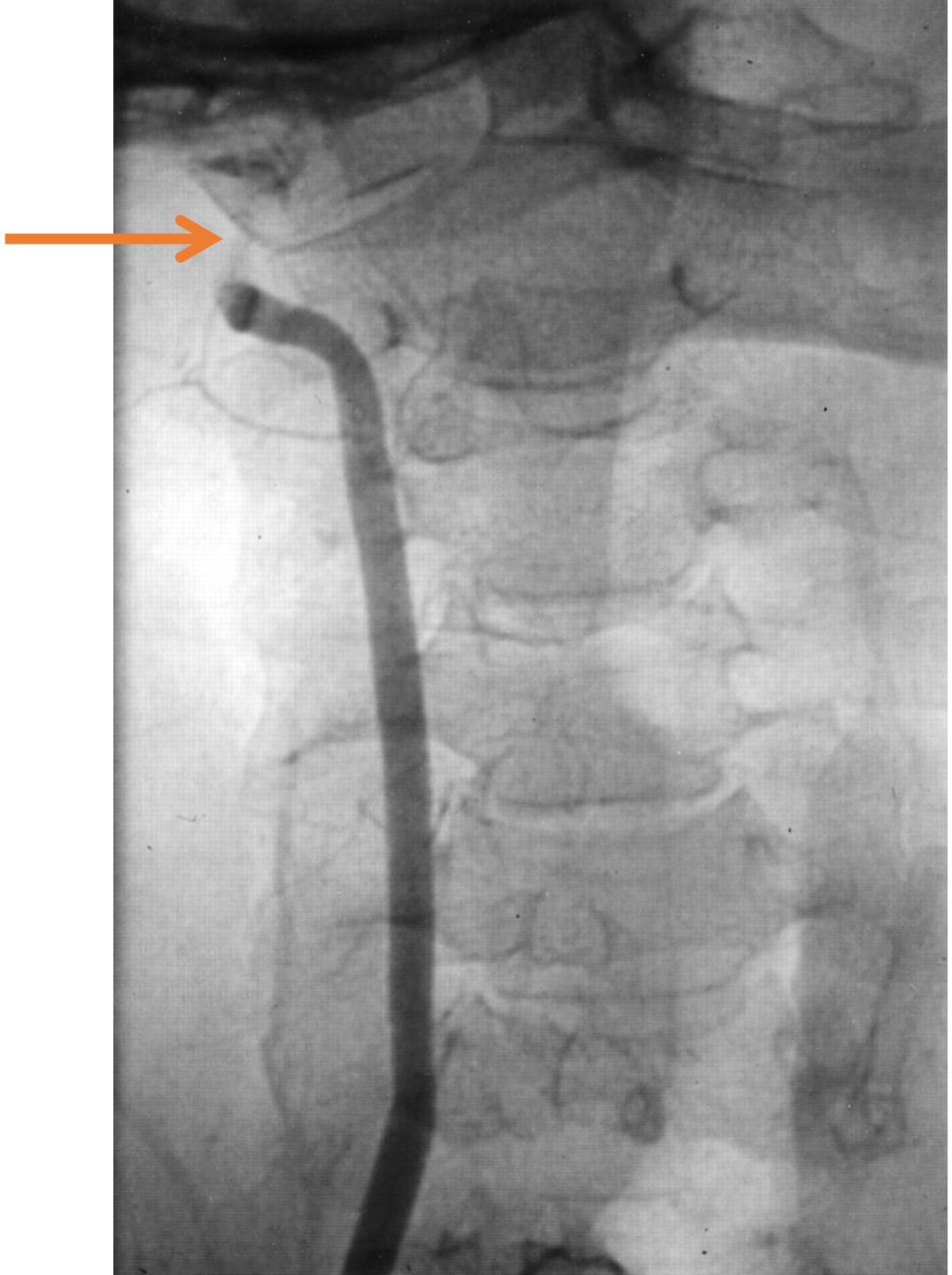
2D video nystagmography (VNG)

Horizontal component toward the right



Clockwise torsional downbeat nystagmus





DSA:

1. Left VA hypoplasia and end in PICA
   - Left Vo 60 ~ 70% stenosis
2. Complete occlusion of right VA at the C2 level after rotation of head toward the left.

Surgical intervention:

- Decompression of right VA from C2 up to its penetration through the dura.
- End-to-side anastomosis of the left VA with the left carotid artery after 3 months (infarction risk)

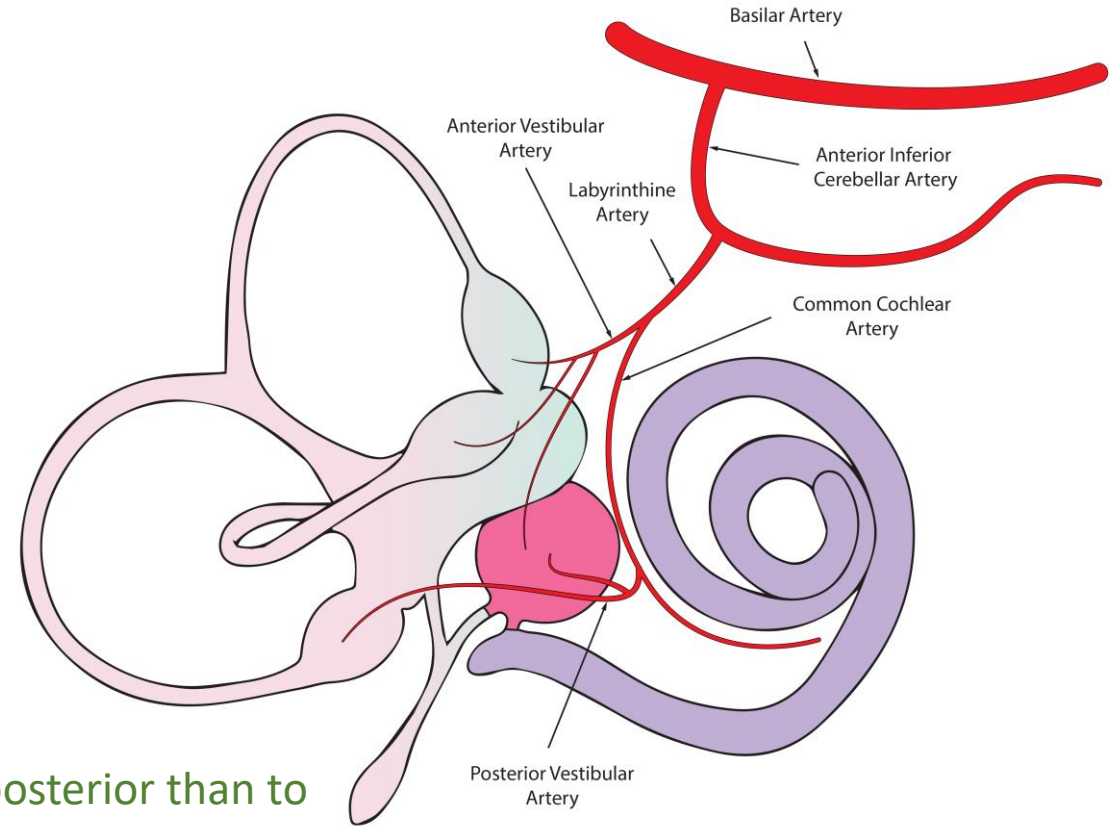
Outcome

- Complete free of symptoms

M Strupp et al. Neurology. 2000 Mar 28;54(6):1376-9.

Transient ischemia of the brainstem or labyrinth?

- Spinning-linear downbeat nystagmus + initial horizontal component toward the compressed VA
  - A transient ischemia of the labyrinth on the side of the compressed artery (Anterior and horizontal SCC)
  - Cochlea → associated tinnitus



More collaterals (intraosseous branches) to the posterior than to the anterior labyrinthine artery→ spare the posterior SCC

Hypo- or hyperfunction of the labyrinth?

- Initially, an excitation by depolarization (causing transient hyperexcitability with ectopic discharges).
  - Positive symptoms of nystagmus and tinnitus
- If ischemia lasts longer, the membrane potential depolarizes further, and the axonal membrane can no longer be excited.
- In severe cases, ischemia of the brainstem will certainly cause other additional central signs and symptoms.

Diagnosis

- Transcranial Doppler sonography
- Digital dynamic angiography → gold standard

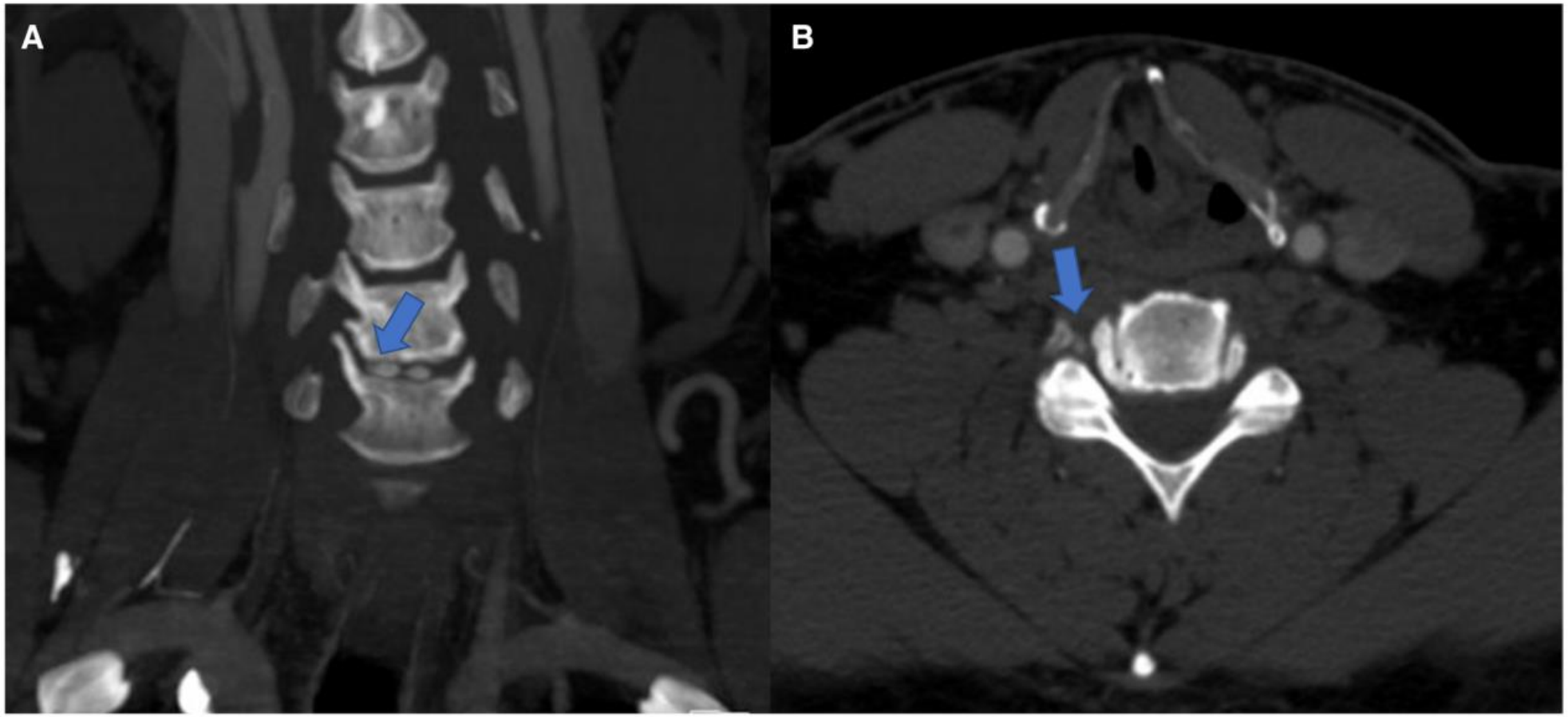
Differential diagnosis

Table 1 Differential diagnosis of cervical vertigo: vertigo, unsteadiness, or oscillopsia triggered/aggravated by head-neck movements

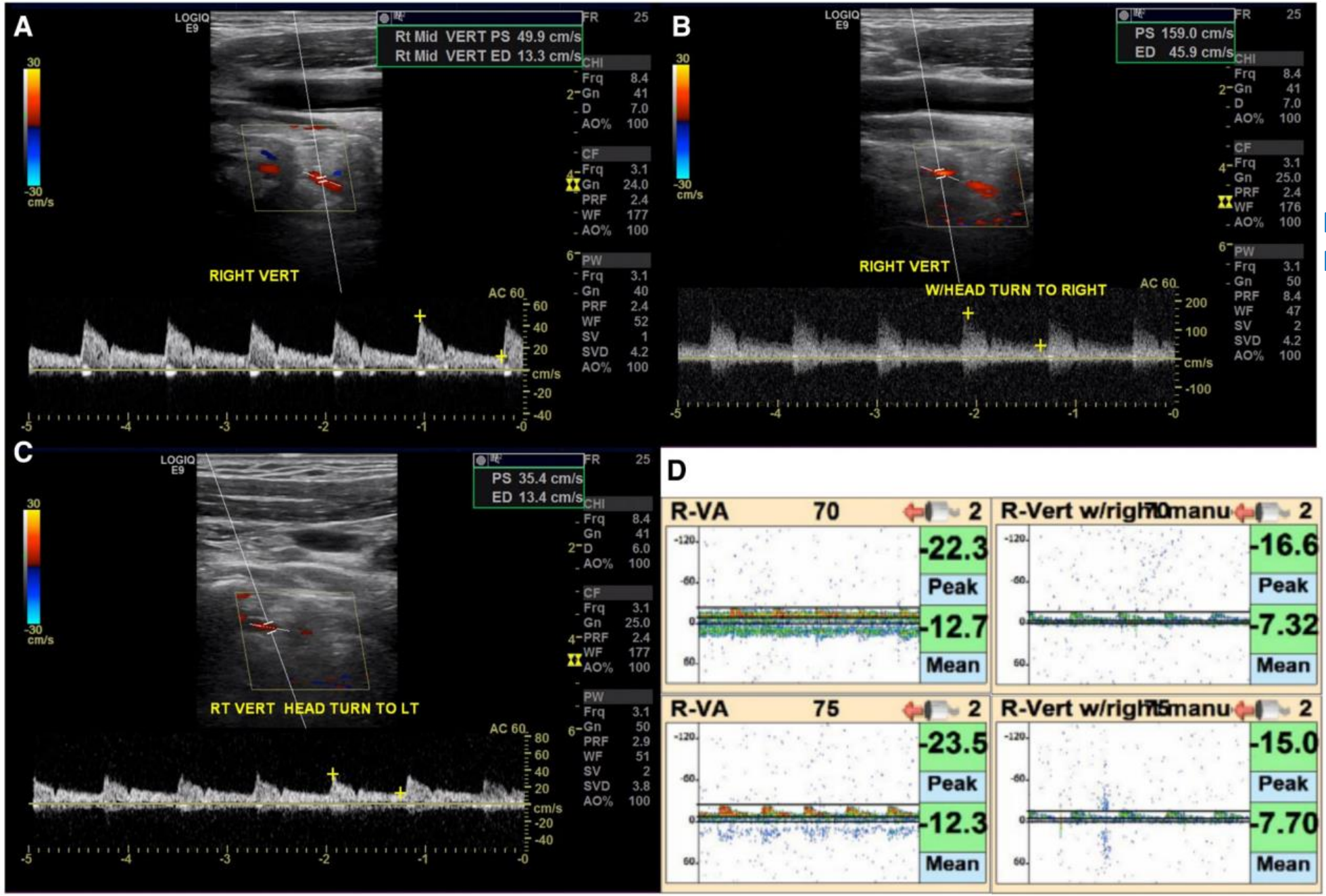
Disorder	*Assumed mechanism*
Labyrinthine:	
Benign paroxysmal positional vertigo	Canalolithiasis, cupulolithiasis
Post-traumatic otolith vertigo	Dislodged otoconia, causing unequal heavy load on macula
Perilymph fistula	Floating labyrinth
Vestibular nerve:	
Unilateral vestibular failure (eg, vestibular neuritis)	Cross coupling effects with acute vestibular tone imbalance
Bilateral vestibular failure	Defective vestibulo-ocular reflex
Vestibular paroxysmia	Neurovascular cross compression
Nerve compression by cerebellopontine angle mass	Conduction block or ectopic discharges
Ocular motor:	
Extraocular eye muscle or gaze paresis	Inappropriate vestibulo-ocular reflex
Central vestibular:	
Central positional nystagmus/vertigo	Cerebellar disinhibition
Migraine without aura	Motion sickness due to sensory hyperexcitability
Migraine with aura (basilar migraine, vestibular migraine)	Spreading depression involving vestibular structures
Vestibulocerebellar ataxia	Vestibulocerebellar dysfunction
Vascular:	
Rotational vertebral artery occlusion	Ischaemic depolarisation
Carotid sinus syndrome	Global cerebral ischaemia
Intoxication:	
Positional alcohol nystagmus/vertigo	Cerebellar and specific gravity differential between cupula and endolymph (buoyancy mechanism)
Drugs (eg, antiepileptics)	Cerebellar and ocular motor

JNNP 2001;71:8–12

A 58 years-old man with dizziness when turning his head to right



Stroke. 2022;53:e26–e29.



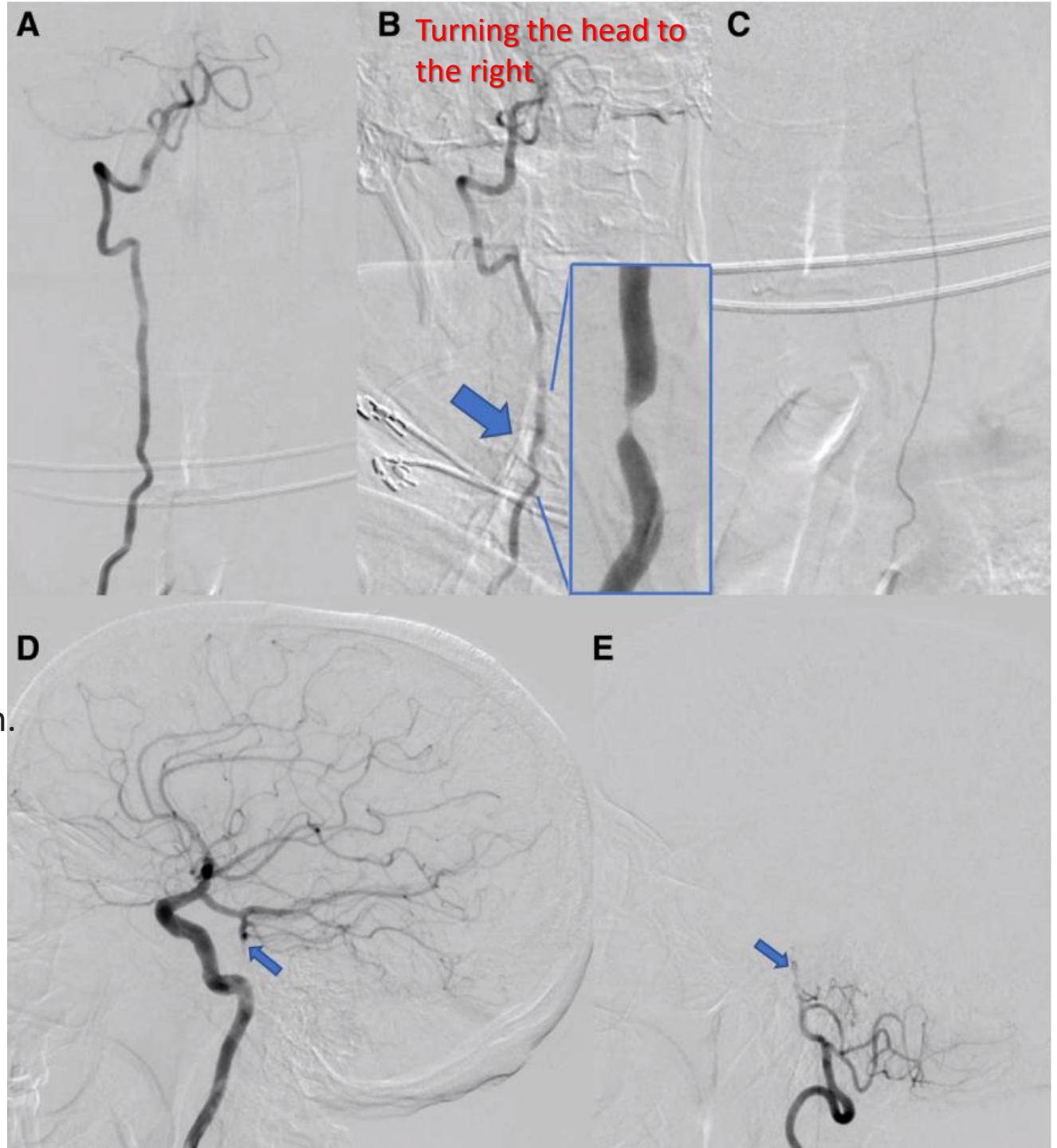
Right head turn
Right VA ↑PSV

Right head turn
Rt intracranial
VA ↓PSV

Left head turn: No effect

Stroke. 2022;53:e26–e29.

Right VA unremarkable



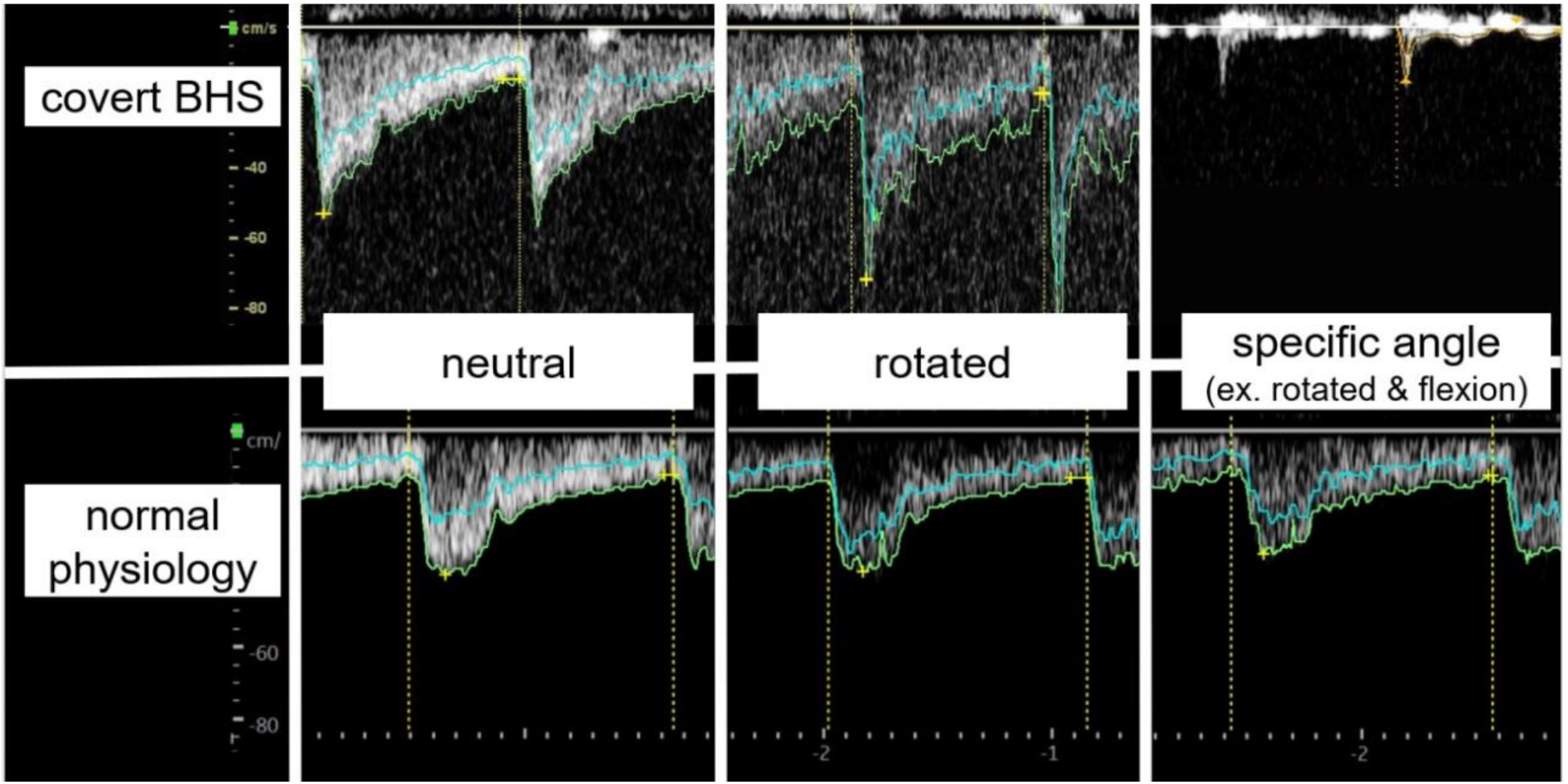
Left VA hypoplasia

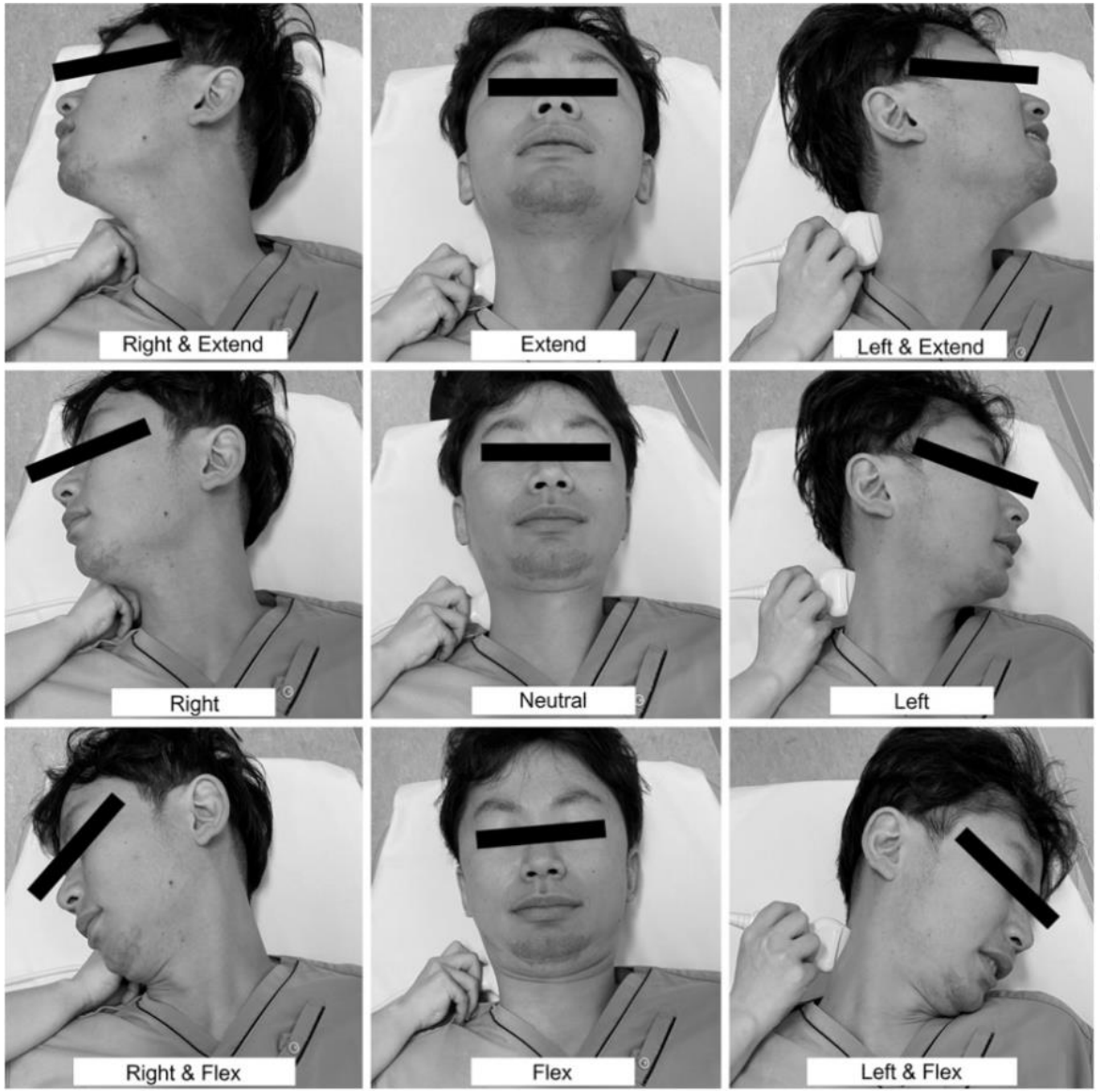
Robust right Pcom.

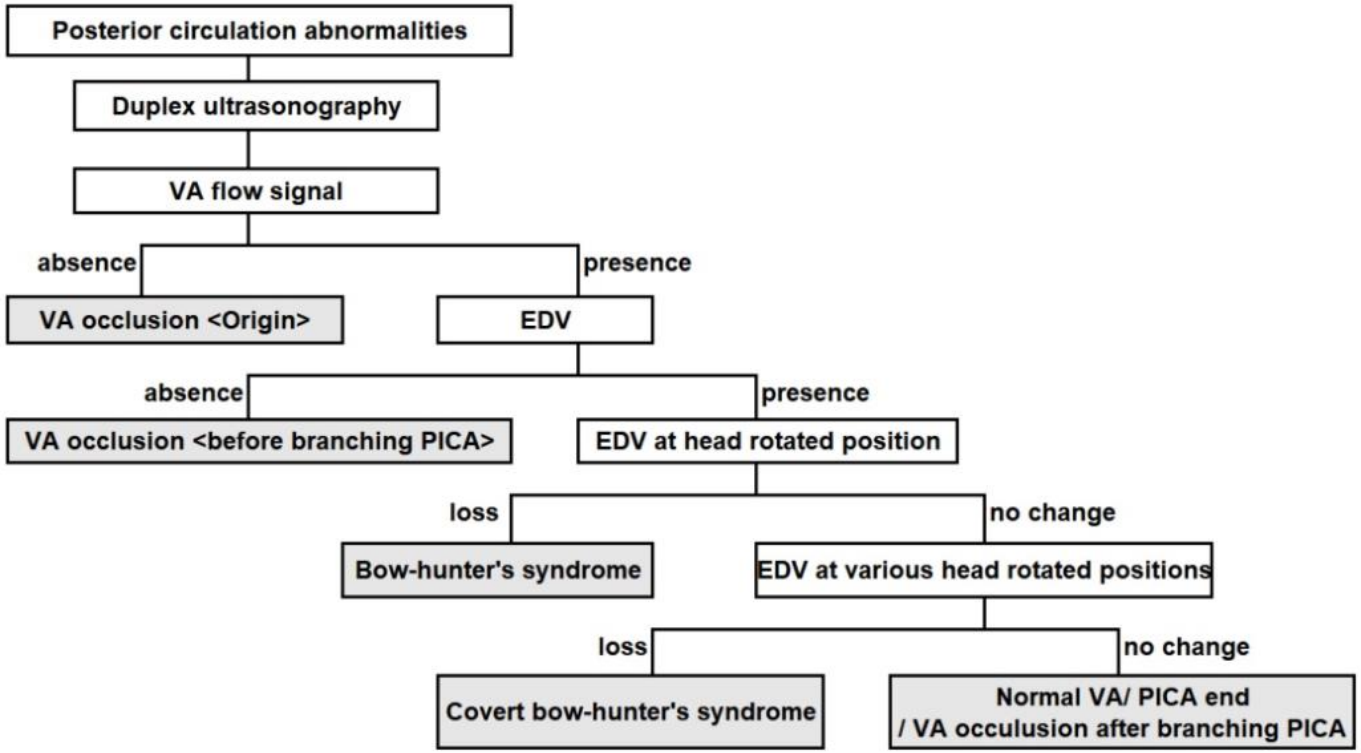
Chronic-appearing mid-basilar occlusion → Supply by ant. circulation

Stroke. 2022;53:e26–e29.

Covert Bow Hunter's syndrome




Right & Extend
Extend
Left & Extend
Right
Neutral
Left
Right & Flex
Flex
Left & Flex


Posterior circulation abnormalities
Duplex ultrasonography
VA flow signal
absence
presence
VA occlusion <Origin>
EDV
absence
presence
VA occlusion <before branching PICA>
EDV at head rotated position
loss
no change
Bow-hunter's syndrome
EDV at various head rotated positions
loss
no change
Covert bow-hunter's syndrome
Normal VA/ PICA end
/ VA occulusion after branching PICA

Treatment

- Conservative treatment
  - Antithrombotic medications
  - Behavioral modification
- Surgical interventions
  - Bony compression of the VA
  - Anterior vs. posterior decompressions
    - The risk of injury to the VA is possibly lower in anterior decompression
- Endovascular interventions

Thank you.